Approach to Neuroimaging

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KEYWORDS

• Computed tomography • MRI • Angiography • Perfusion imaging • Stroke

KEY POINTS

- Non-contrast CT has a low sensitivity for certain neurologic emergencies including stroke and overreliance may lead to misdiagnosis.
- Understanding the strengths, limitations, sensitivity and specificity of various neuroimaging modalities can improve diagnostic accuracy and reduce misdiagnosis.
- A systematic approach to imaging interpretation is paramount for the emergency medicine clinician for timely, thorough and consistent diagnosis.

INTRODUCTION

When it comes to the diagnosis and management of neurologic emergencies in the emergency department (ED), timely access to detailed imaging of the brain, cerebral vasculature, and spine is the key. Evolution in both the understanding and treatment of neurologic emergencies, such as traumatic brain injury, ischemic and hemorrhagic stroke, transient ischemic attack, reversible cerebral vasoconstriction syndrome, and cerebral sinus thrombosis, has underscored the importance of early and appropriate neuroimaging in the emergency setting. Not surprisingly, ED neuroimaging utilization is on the rise. Between 2007 and 2017 neuroimaging per 1000 ED visits rose by 72% with computed tomography (CT) and CT-angiography (CTA) leading this increase.[1] Despite this, studies have shown that emergency medicine (EM) clinicians are likely deficient in their ability to interpret these studies.[2,3] This increase in neuroimaging utilization has led to initiatives, such as the American College of Emergency Physicians (ACEP) "choosing wisely" campaign and outlines the importance for EM clinicians to understand the strengths and weaknesses of the different neuroimaging modalities, as well as the importance of an organized and systematic process for ordering, evaluating, and interpreting these studies.

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Abbreviations	
ED	emergency department
CT	computed tomography
CTA	computed tomography angiography
EM	emergency medicine
NCCT	non-contrasted computed tomography
MCA	middle cerebral artery
SAH	subarachnoid hemorrhage
DAI	diffuse axonal injury
ICH	intracranial hemorrhage
IPH	intraparenchymal hemorrhage
MRA	magnetic resonance angiography
ASL	arterial spin labeling
RF	radiofrequency
CTP	perfusion computed tomography
CBV	cerebral blood volume
CBF	cerebral blood flow
MTT	mean transit time
TTP	time to peak
SWI	susceptibility-weighted imaging
FLAIR	fluid-attenuated inversion recovery
DWI	diffusion-weighted imaging
LVO	large vessel occlusion

Computed Tomography

A non-contrasted CT (NCCT) scan is the foundation of emergent neuroimaging. CTs are widely available, quickly obtained, relatively safe, and lack any hard contraindications. Additionally, NCCT has a high sensitivity and specificity for many of the critical diagnosis that are pertinent to ED care, such as in traumatized patients.[4]

CT images are cross-sectional, computer-generated reconstructions created by the analysis of the attenuation of radiograph beams that are passed through a patient at desired widths and picked up by multiple detectors placed 180° opposite of the radiograph beam source, which spin around the patient at a rapid rate. Using a modern generation scanner, an NCCT of the head can be completed in less than 10 sec.[5] The attenuation of the radiograph beams are calculated as projection images based on the density of different tissues through which the beams pass. Higher attenuation results in images with higher density, which appears whiter on the scan. Areas of lesser radiograph attenuation appear dark (this occurs in soft tissues and when the beam encounters air). The attenuation coefficient in CT imaging is measured in Hounsfield units on a scale of −1000 (least dense *e.g. air*) to +1000 (most dense e.g. bone).[5,6]

It is important for EM clinicians to have a systematic approach to evaluating CT scans in order to rapidly identify pathology that may alter a patient's care plan. One well-known method for systematizing the evaluation of a cranial CT is following the "Blood Can Be Very Bad" mnemonic.[3] In this method, the physician evaluates the scan first by looking for the presence and location of blood (Blood), as well as the effect that it is having on the surrounding brain. Next, evaluate the "cisterns" (Can) with particular focus on the suprasellar, quadrigeminal, peripontine, and infrapontine cisterns in the midbrain region (**Fig. 1**).

Closure of the circummesencephalic cistern is one of the earliest signs of increased intraranial pressure (ICP) in the setting of subarachnoid hemorrhage (SAH).[7] Additionally, the Sylvian cistern is important as it represents the "end organ" portion of the middle cerebral artery (MCA) and will be one of the first areas to lose gray/white

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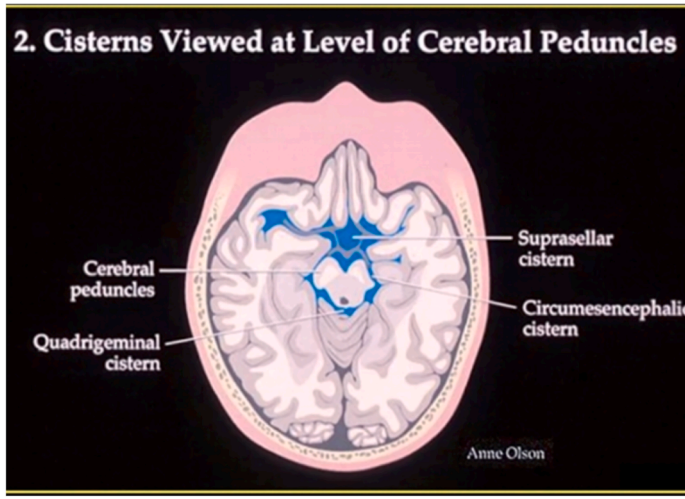

Fig. 1. Cisterns viewed at level of cerebral peduncles. (*Image Courtsey* by Dr Andrew Pellet.)

differentiation in the setting of MCA territory ischemia (this is called insular ribbon sign), as well as being a common sight of SAH from aneurysm (Fig. 2).

Brain (Be), which should be evaluated for symmetry, to ensure no effacement of the sulci/gyri and for a grossly symmetric shape, preservation of the grey-white differentiation, and for midline shift. Ventricles and vessels (Very) refers to the need to assess the ventricles for effacement, blood, enlargement, or shift. The vessels should be evaluated for hyper-dense vessels, which indicate clot presence, this is a specific but not sensitive finding for vessel occlusion. The presence of clot should always be compared to the patient's physical examination to ensure that the suspected neurologic deficit when a clot is identified is in fact congruent with the patient's presentation. Lastly, bones (Bad) reference the need to evaluate the skull for fracture. It is important

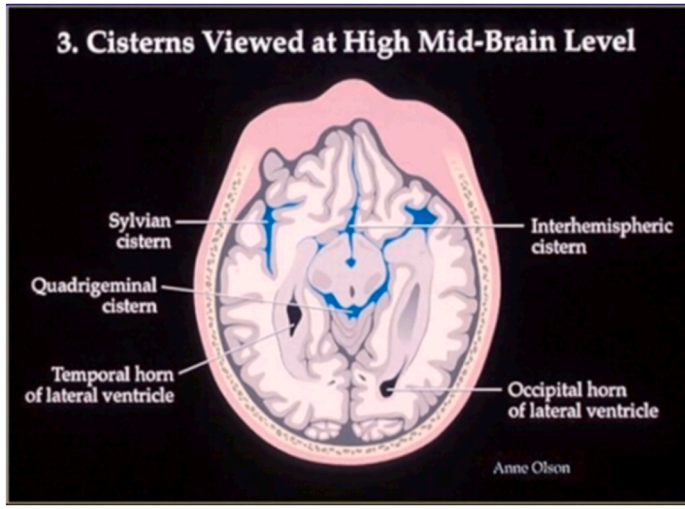

Fig. 2. Cisterns viewed at high mid-brain level. (*Image Courtsey* by Dr Andrew Pellet.)

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to remember that the hypo-densities that are identified with skull fracture will only be obvious when there is a displaced fracture and that any lucency in the bones should be compared to the contralateral side to ensure that it is not a suture line. Because linear, non-displaced fractures can be hard to identify, it is also important to look for indirect signs of a skull fracture including the presence of fluid in areas, such as the mastoid air cells, or in the maxillary, ethmoid or sphenoid sinuses, as well as the presence of air inside the cranium as this indicates a disruption of the bone and dura that allowed the air to enter the subdural space (**Table 1**).[3,7–10]

If all of these structures have been evaluated and are without abnormality, then the NCCT is not providing any emergent diagnosis. The clinician should consider if further investigation with MRI is necessary or if there are alternative explanations for the patient's symptoms (**Fig. 3**).[7,8]

The NCCT is usually the most important neuroimaging modality in the setting of trauma.[11] Traditional assessment tools, such as Glascow Coma Scale (GCS), have been verified to have great interobserver reliability; however, they fail to identify the extent, nature or location of injuries that lead to clinical deficits. Expedited neuroimaging therefore plays an important role in providing timely information that can dramatically change patient management by rapidly identifying reversible injuries that require urgent intervention.[10–12]

The latest generation of CT scanners is highly sensitive and specific for almost all traumatic and trauma-related injuries with the exception of Diffuse Axonal Injury (DAI).[4] MRI is typically considered a secondary imaging modality due to time that it takes for image acquisition, especially in a potentially unstable patient population.[11] MRI is also highly prone to image degradation from motion artifact requiring patients to remain still for lengthy periods of time, which may prove logistically difficult secondary to pain or mental status. Lastly, due to the powerful magnetic fields created by an MRI machine, they may be incompatible with much of the other trauma-related equipment, or metallic foreign bodies, and they are largely less available than CT scanners.

The injury patterns that are identified on NCCT in traumatic brain injury (TBI) patients are separated into 2 categories. Primary injuries are those caused by direct impact to the head including: skull fractures, hemorrhage, contusion, and DAI. Secondary injuries are often the sequela of primary injuries and include: edema, herniation, and chronic traumatic encephalopathy.

In the setting of suspected head trauma, one of the most important early questions to address is whether or not there is evidence of intracranial hemorrhage (ICH). NCCT is an excellent tool for identifying brain contusion particularly when it presents with a hemorrhagic component. The attenuation of blood on NCCT is due to hemoglobin content. Anemic patients have less change in attenuation and NCCTs are at increased risk for missing areas of hemorrhage.[13] Studies have shown that there is a linear relationship between the density of blood on NCCT and a patient's hemoglobin level. Notably, when the patient has hemoglobin between 8 and 10 g/dL acute subdural hematoma can appear iso-dense with cerebrospinal fluid (CSF).[13,14]

There are 4 primary kinds of hemorrhage that ED clinicians should be aware of when evaluating an NCCT of a trauma patient (epidural, subdural, subarachnoid, and intraparenchymal). Epidural hematomas (EDHs) classically appear as convex, "lens shaped" hyperdensities, which do not cross suture lines on the skull. EDH almost always occur in the setting of an associated skull fracture (>90%), and are the consequence of arterial bleeding (90% – 95%) rather than venous bleeding into the space outside the dura mater.[4] The bleeding source is classically the middle meningeal artery and only 50% of EDH's are associated with the classic "lucid period" on history. Since the blood is outside the dura, this means that EDH are very amenable to surgical

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Table 1
Description of the 'Blood Can Be Very Bad' mnemonic

Mnemonic	Structures Evaluated	Imaging Findings to Identify	Clinical Pearls
Blood	Evaluate for presence of blood	Identify location of hyperattenuating lesion, location and mass effect	• Epidural Hemorrhage. • Subdural Hemorrhage. • Subarachnoid Hemorrhage. • Intraparenchymal hemorrhage.
Can	Cisterns	Evaluate for closure or compression of the cisterns	• Suprasellar, Quadrigeminal, Peripontine and Infrapontine cisterns these are in the midbrain region and are high yield. • Closure of circummesencephalic cistern is one of the earliest signs of increased ICP. • (Insular ribbon sign): The Sylvian cistern is important as it represents the "end organ" portion of the middle cerebral artery (MCA) and will be one of the first areas to lose gray/white differentiation in the setting of MCA territory ischemia.
Be	Brain	Symmetry	• Evaluate for midline shift, general symmetry and preservation of the grey-white junction.
Very	Ventricles/Vessels	Effacement, shift, dilation, hyperdense vessels	• Effacement of the ventricles indicates some mass effect or increased ICP. • Dilation of the ventricles could indicate hydrocephalus and should prompt inquiry into the cause. • A hyperdense vessel is a specific finding for clot presence but not sensitive.

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Table 1
(continued)

Mnemonic	Structures Evaluated	Imaging Findings to Identify	Clinical Pearls
Bad	Bones	Evaluate for symmetry of the skull bones, as well as the sinuses for opacification, always compare findings to the contralateral side	• Any presence of air inside the dura matter suggests a skull fracture in the context of trauma. • Opacification of the mastoid air cells or other sinuses in the correct clinical context is an indirect sign of skull fracture.

decompression as the underlying brain is often intact and patients will improve significantly if the mass effect of the accumulating blood is removed prior to herniation (**Fig. 4**).

Subdural hematomas (SDH) typically appear as crescent-shaped, convex, blood accumulations that cross suture lines. SDH's are the consequence of venous bleeding from the bridging veins between the arachnoid and dura matter and can occur from much lower energy mechanisms than other forms of traumatic ICH. These are commonly found along cerebral convexities: frontoparietal, falx cerebri, and tentorium cerebelli and rarely the posterior fossa. In contrast to other forms of ICH, SDH poses a diagnostic challenge as it can appear hypodense, iso-dense, or hyperdense on CT when compared to the surrounding cerebrospinal fluid depending on the timing and staging of the hematoma. Acute SDH is commonly hyperdense, except in the setting of anemia (hgb 8 – 10 g/dL) in which case they can appear iso-dense when compared to the cortex of the brain.[4,13] Subacute subdural bleeds appear isodense or hypodense (darker) when compared to the cortex. Diagnostic clues on CT are to look for inward displacement of CSF, mass effect (effacement of the sulci adjacent to the collection), compression of the ipsilateral ventricles, and midline shift. Follow-up imaging with a contrasted CT in order to evaluate for inward displacement of cortical

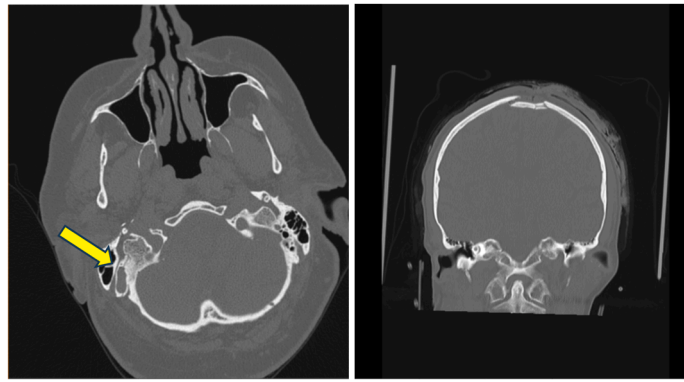

Fig. 3. Left: Subtle fracture through right jugular foramen with adjacent mastoid air cell effusion Right: Depressed skull fracture on bone window. The yellow arrow points to the subtle fracture described.

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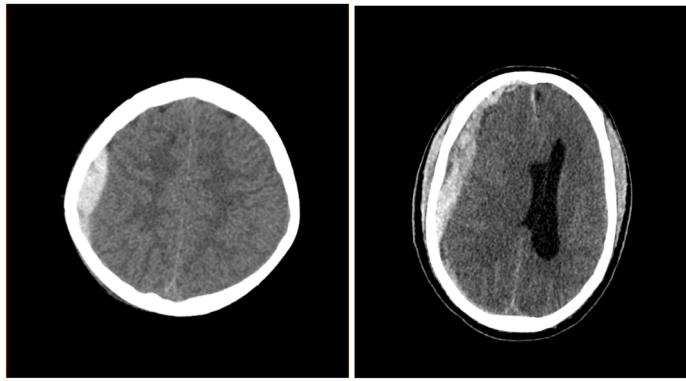

Fig. 4. Left: Epidural hematoma appearing over the right temporal/parietal region Right: Acute Subdural hematoma over the right hemisphere with midline shift and effacement of the right lateral ventricle.

vasculature or MRI may be necessary to further characterize iso-dense consolidations suspicious for SDH (Fig. 5).

SAH occurs from blood accumulation between the pia and arachnoid matter and commonly presents as hyperdensities along the cortical sulci. In traumatic SAH, these will be typically seen near the site of impact (coup) or as accumulations in the cisterns near the midbrain. SAH should also be considered outside of traumatic injury in the setting of sudden onset severe headache. In this context, greater than 85% of SAH is secondary to an underlying aneurysm rupture with the majority originating from the Circle of Willis.[15] This means that the hypodensities can be seen on CT in the suprasellar or central basal cisterns and can have diffuse peripheral extensions. In the case of non-traumatic SAH, CTA or magnetic resonance Angiography (MRA) would be indicated to evaluate for the source of bleeding (Fig. 6).

Intraparenchymal hemorrhage (IPH) can be seen in the setting of trauma, but is often spontaneous and accounts for approximately 10% of stroke presentations.[14] The NCCT is a crucial aspect to any stroke workup as it is paramount to rule out a

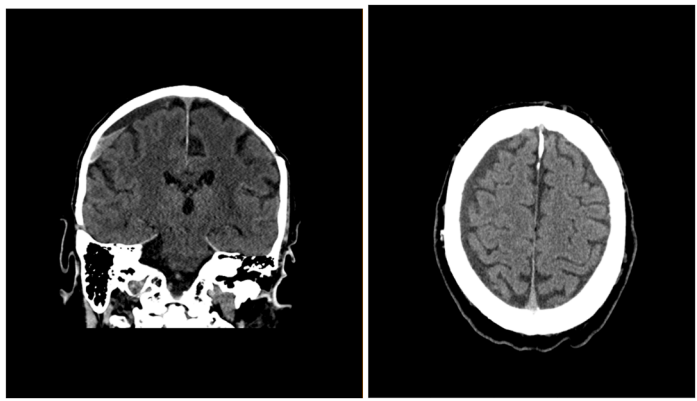

Fig. 5. Left: Layering subdural hematoma over the right cortex with various densities of blood from multiple bleeding events Right: Subacute subdural collection over right hemisphere appearing hypodense.

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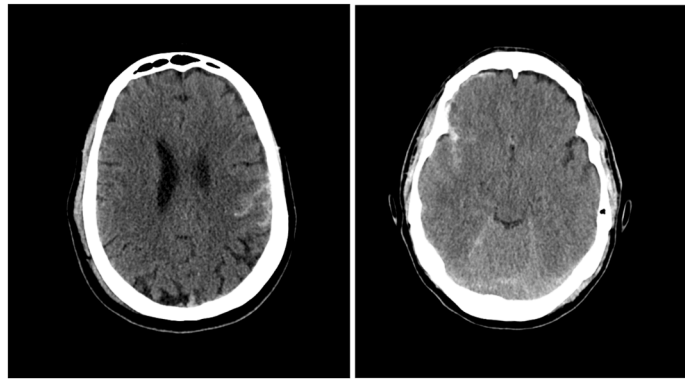

Fig. 6. Left: Subarachnoid hemorrhage over the left frontal lobe Right: Subarachnoid hemorrhage involving the right sylvian fissure.

hemorrhagic stroke as hemorrhage is a contraindication to thrombolysis. IPH presents on NCCT as a hyper-attenuating lesion within the brain parenchyma and should be observed for not only the location of the bleed, but the effect that it is having on surrounding tissue including signs of local or more diffuse mass effect (effacement of sulci) or for signs of herniation.[15,16] IPH is commonly the consequence of longstanding hypertension and the resultant smooth muscle hyperplasia of cerebral arterioles. However, other common causes include vascular malformations, bleeding intracranial masses, vasculitis/vasculopathy, and hemorrhagic conversion of ischemia.[14] CT or MR angiography can be used in the setting of IPH to better characterize the underlying etiology of the bleed and aid in predicting early hematoma expansion. The presence of intraventricular hemorrhage tends to be a poor prognostic factor (**Fig. 7**).

While far less sensitive than MRI for brain ischemia and space occupying lesions, NCCT is still generally considered the most appropriate first-line imaging modality in patients presenting with acute neurologic deficits. Contrast-enhanced CT may be preferred in circumstances when a brain abscess is being considered.

In addition to rapidly ruling out hemorrhagic etiologies of neurologic deficits with an NCCT, there are many instances when advanced imaging of the cerebrocephalic

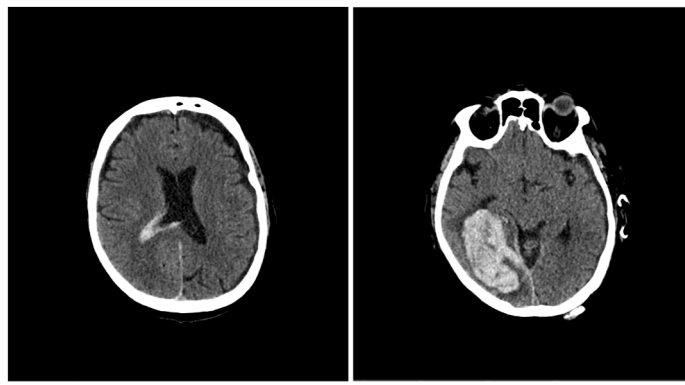

Fig. 7. Left: Intraventricular hemorrhage in posterior horn of right lateral ventricle Right: Large right sided intraparenchymal hemorrhage with mass effect.

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vasculature is necessary as blood flow disruption whether by thrombosis, embolism, external compression, vessel disruption or dissection leads to decreased peripheral perfusion, and rapid neuronal cell death and lasting neurologic deficits. Vessel imaging therefore plays a crucial part in the assessment of suspected stoke patients, as well as those with penetrating neck trauma or high-mechanism blunt head and neck injuries when a blunt cerebrovascular injury may be present. Additionally, venous imaging with CT venogram is often the first-line imaging for patients with suspected cerebral venous thrombosis as it is more readily available, and CT imaging can be obtained more quickly even though MRI is more sensitive for CVT (**Fig. 8**).[17,18]

Angiography, Computed Tomography Angiography, and Magnetic Resonance Angiography

The gold standard of vessel imaging is direct angiography. However, this is typically the highest morbidity imaging method due to the inherent risks that come with instrumenting the blood vessels.[4] CT and MRI vessel imaging serve as the primary non-invasive modalities available to ED clinicians for imaging the intracranial and extra-cranial vasculature when vessel occlusion or injury is suspected.

CTA is obtained by timing a CT scan with a bolus of intravenous (IV) contrast such that the contrast highlights the anatomy of the blood vessels during the scan. These images can be timed to obtain venous or arterial phases such that the anatomy and patency of arterial system or the veins can be evaluated respectively. Because CTA images the vessels while they are filled with a contrast medium, they can be understood to be directly evaluating the anatomy of the vessel. This is in contrast to MRA and magnetic resonance venography, which are capable of imaging cerebral vasculature in the absence of contrast administration through a variety of different techniques including Time of Flight angiography, black blood sequencing, and arterial spin labeling (ASL).

Non-contrasted Magnetic Resonance Angiography

In patients unable to receive IV contrast agents due to allergy or renal impairment, non-contrasted MRA can provide an alternative for accurate vascular imaging. MRA sequences can accentuate the differences in magnetization between stationary tissue and fresh flowing blood entering the imaging plane, which has not been exposed to the magnetizing radiofrequency (RF) pulse used to generate tissue polarization.

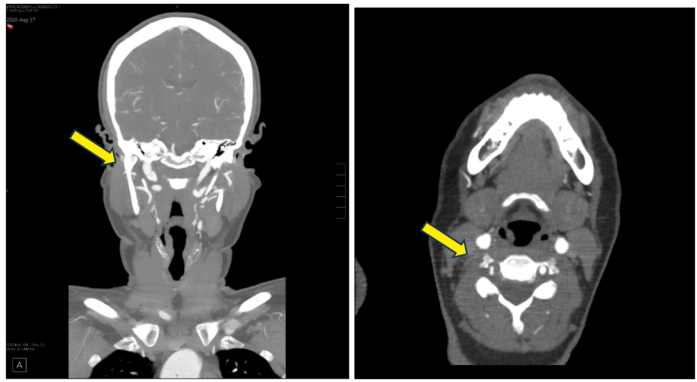

Fig. 8. Left: CTA showing right ICA dissection and pseudoaneurysm formation Right: CTA showing right vertebral artery dissection.

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Vasculature will appear intense/bright on imaging compared to the surrounding tissue. These image slices can be stacked and reconstructed to generate a 3-dimensional image of the vascular structures of interest. Though limitations may exist in identifying smaller vasculature, non-contrast MRA is highly sensitive in identifying large vessel occlusions (LVO), stenosis, ateriovenous (AV) malformations, and aneurysmal disease.

When considering a stroke patient with ischemic etiology, obtaining non-invasive angiography of the head and neck is an important part of the neuro imaging assessment as the location and extent of clot burden directly impacts the management that our consultants might recommend for these patients. For example, clots greater than 8 mm in length have a low likelihood of achieving reperfusion through thrombolysis alone.[16,17] These patients may be better candidates for mechanical thrombectomy, which in many cases may require rapid identification and patient transfer in order to meet set time criteria for which interventions have been proven to improve outcomes.

Perfusion Imaging

Perfusion CTs (CTP) utilize dynamic imaging techniques to help distinguish areas of tissue at risk of infarction following an ischemic event, referred to as the "ischemic penumbra", from areas which are no longer salvageable with restored perfusion, referred to as the "core infarct".

After a bolused injection of an IV contrast agent, rapid-sequence CT imaging can be obtained as the agent passes through the region of interest. By measuring the migration of contrast as it enters and eventually exits the region of interest, cerebral blood volume (CBV), cerebral blood flow (CBF), mean transit time (MTT), and time to peak (TTP) flow can be derived.

Typically, areas with low CBF, low CBV, and a prolonged MTT or TTP, indicate core infarct tissue unlikely to be salvaged. Regions with preserved CBV but reduced CBF and prolonged MTT or TTP suggest penumbral tissue. A mismatch between these 2 regions can guide therapeutic decisions, such as thrombectomy or thrombolysis, as interventions are most effective when targeting salvageable penumbra tissue. A patient with a large area of penumbral tissue surrounding a small area of core infarct may be more likely to respond to therapies, such as thrombolysis or thrombectomy, than a patient with a large core infarction.

In facilities where MRIs can be rapidly obtained, MR perfusion may be an alternative to CTP, which avoids ionizing radiation. Both are able to show ischemic penumbra and core infarct. Dynamic susceptibility contrast MRI, requires administration of a gadolinium contrast agent and rapid acquisition of MR images. Similar to CTP, it is able to determine CBV, CBF, MTT, and TTP by measuring changes in MR signal intensity due to passage of contrast. ASL is another perfusion technique, which does not rely on contrast administration. Both may be preferred when aiming to minimize radiation exposure (e.g. younger patients).

While CTP studies are an imperfect tool, the combination of these studies along with CTA and non-contrasted CT has been shown with some protocols to be able to identify both the ischemic core and the penumbra. EM clinicians should know about the existence of these scans as they may be used by some specialists to guide management decisions in some circumstances when MRI is not available. It is also important to understand that for many patients presenting to the ED for stroke it is not, in fact, necessary to determine penumbra volume as patients within the time window for endovascular intervention with LVO and small infarct cores almost all warrant treatment.

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Imaging The Emergency Department Patient with New-onset Seizure

First-time seizure is another common indication for ED neuroimaging. American Academy of Neurology guidelines recommend immediate NCCT in patients presenting to the ED with first time seizure. In patients with known seizure disorder and typical presentation for their seizure pattern, CT is less likely to change management. However, according to some studies, CT imaging in the ED will change the management of first time seizure in 9% to 17% of patients according to some evidences.[19] In children, CT will change management 3% to 8% of the time, and in children <6 mo it can change management in up to 50% of cases.[19] This is especially important in immunocompromised patients and those with toxoplasmosis. Change in neurologic examination, strong historic features, or focal seizure onset are predictive of an abnormal CT. Some of the CT findings that lead to acute changes in management include: TBI, depressed skull fracture, subdural hematoma, non-traumatic bleed from arteriovenous malformation (AVM) or other cerebral hemorrhage, cerebrovascular accident (CVA), tumor, brain abscess, cysticercosis, obstructive hydrocephalus, ventriculoperitoneal (VP) shunt malfunction, Aicardi syndrome, Miller-Diecker Syndrome, Tuberous Sclerosis, and Toxoplasmosis. In the setting of a normal initial NCCT in patients with persistently abnormal mental status or evidence of ongoing focal seizure activity, a gadolinium-enhanced contrast MRI should be obtained to rule out other structural causes. It is important to note that CT has poor sensitivity for low-grade gliomas, hippocampal sclerosis, or cavernous malformations.

MRI

MRI, while more difficult to obtain emergently than NCCT, can be a valuable diagnostic imaging modality for utilization in the ED. Its resolution in central nervous system (CNS) imaging is in general superior to that that of its CT counterpart, particularly in the realm of early stroke detection, posterior circulation strokes, stroke mimics (e.g. multiple sclerosis), hypoxic ischemic encephalopathy, DAI, and late or small SAH.[20–25]

To generate MR images, the patient is placed in a strong uniform magnetic field (B_0) that aligns the nuclear spins (i.e. magnetic moments) of hydrogen atoms in the body with B_0. The alignment and precession of hydrogen's magnetic moment with B_0 is then intentionally disrupted by transient application of a RF pulse with an external magnetic field component (B_1) in a plane orthogonal to B_0. This causes the hydrogen spins to precess in phase with one another and in alignment with the B_1 field resulting in a net magnetic moment aligned with the B_1 plane.[26,27]

Once the B_1 pulse is removed, the net magnetization of nuclei in the B_1 plane gradually diminishes as individual hydrogen nuclei lose precessional phase coherence. This loss of magnetization in the B_1 plane is referred to as T2 decay and occurs at a rate that varies depending on the tissue or fluid in which the hydrogen atoms are bound (with T2 relaxation times [e.g. time to decay] following the general trend: CSF> gray matter> white matter). By timing data acquisition appropriately, MRI can generate contrast between tissues based on intrinsic differences in T2 relaxation.[26,27]

Simultaneously while hydrogen nuclei are losing polarity in the B_1 plane, net magnetization is being restored in the B_0 plane as this field is being applied at all times. Restoration of hydrogen nuclei's alignment with the B_0 magnetic field is referred to as T1 relaxation and like T2 relaxation, occurs at a rate that is dependent on the tissue or fluid that hydrogen is bound to (with T1 relaxation times following the general trend CSF> gray matter> white matter). Similar to T2 imaging, by timing data acquisition appropriately, MRI can generate contrast between tissues based on intrinsic differences in T1 relaxation.[26,27]

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In essence, T1 and T2 relaxation times govern how tissues respond to the magnetic field and RF pulses used in MRI. The timing, duration, and gradient of external RF pulses in addition to timing of data acquisition, create unique imaging sequences each with their own strengths and weaknesses. Some sequences are more useful in visualizing anatomy while other sequences can more readily illuminate the presence of pathology.[26,27]

A standard brain MRI series will typically include the following image sequences: T1-weighted, T2-Weighted, T2* (and/or susceptibility-weighted imaging [SWI]), fluid-attenuated inversion recovery (FLAIR), diffusion-weighted imaging (DWI).[28,29]

T1-weighted sequences

This sequence primarily emphasizes differences in T1 relaxation times. Typically tissues with a shorter T1 recovery (e.g. fat and white matter) will appear bright/white whereas tissues with a longer recovery time will appear darker (e.g. gray matter) with CSF appearing black. T1 images can be useful for visualizing anatomic structures, delineating gray matter, white matter and CSF, identifying atrophy, as well as the presence of masses.[30]

T2-weighted sequences

This sequence primarily emphasizes differences in T2 relaxation times. Typically tissues with greater water content have longer T2 relaxation times will appear bright (e.g. gray matter) with CSF, comprised mostly of water, being white, whereas tissues with a shorter relaxation times will appear darker (e.g. fat and white matter). T2 sequences are sensitive to fluid content and tissue edema and as such are helpful in detection of pathologic conditions, such as tumors, certain infections, and inflammatory conditions. Typically, the edematous, pathologic tissue will have a brighter appearance compared to surrounding normal parenchyma (**Fig. 9**).[30]

T2* and susceptibility-weighted image sequencing

T2 sequencing relies on the loss of transverse magnetization (magnetization in the B_1 plane) and acquiring data when T2 decay is greatest between tissue layers, allowing for contrast amongst the tissue layers in the final image. In reality, loss of transverse magnetization occurs more rapidly than predicted due to inhomogeneities in the B_0 magnetic field and localized field distortions due to irregularities in the tissue itself (e.g. hemorrhage, calcifications, and iron deposits). This is referred to as T2*. Unlike T2 sequencing, which utilizes compensatory pulses to correct for losses from these irregularities, T2* imaging does not. These field irregularities on T2* imaging will

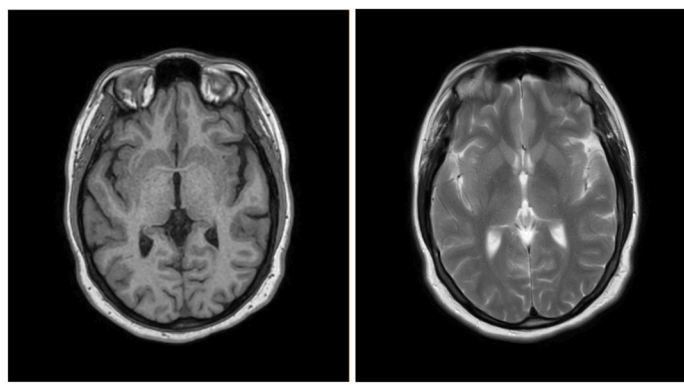

Fig. 9. Left: Normal T1 weighted image, Right: Normal T2 weighted image.

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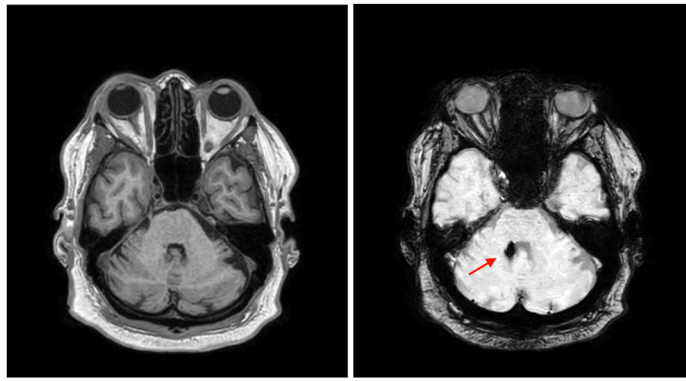

Fig. 10. Left: T1 Weighted image Right: SWI image, same patient, demonstrating cerebellar hemorrhage. The red arrow points to the cerebellar hemorrhage described.

typically appear as dark spots or regions due to localized areas of signal loss. As such, it can be helpful in identifying areas of hemorrhage (due to the paramagnetic properties of blood), AV malformations, and tumors containing microhemorrhages.[31] SWI is a specific MRI technique that uses T2*-weighted imaging but adds advanced post-processing techniques to enhance the visibility of hemorrhage, and deposits of iron or calcium (**Fig. 10**).[32]

Fluid-attenuated inversion recovery

FLAIR is essentially a T2-weighted image that incorporates an inversion recovery pulse sequence to nullify the signal from fluids. White matter will appear darker than gray matter as in a standard T2 image; however, due to the nullification of the signal from fluids, CSF will appear black. This sequence is particularly useful in detection of periventricular abnormalities, which could be difficult to visualize with standard T2 imaging due to CSF's bright appearance, such as SAH, periventricular involvement of multiple sclerosis (MS), and meningitis/encephalitis, when a contrast agent is administered (**Fig. 11**).[33]

Diffusion-weighted imaging

DWI relies on its ability to detect the diffusion of water molecules (or lack thereof) within tissue. It is highly sensitive in detecting restricted diffusive movement of water

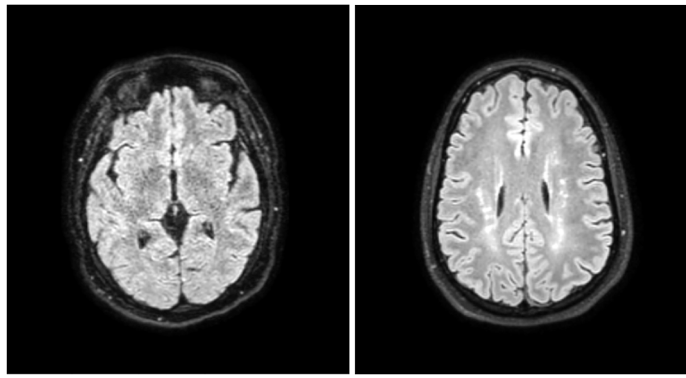

Fig. 11. Left: Normal FLAIR sequence imaging Right: Periventricular FLAIR enhancement in patient with Multiple Sclerosis.

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molecules, which is a common characteristic of ischemic or edematous tissues. In healthy tissues, water molecules exhibit random Brownian motion and can diffuse readily. In conditions causing cytotoxic edema, such as acute ischemia or axonal injury, water diffusion is significantly restricted. This is a result of failure of the Na+/K+ transmembrane pump following ATP depletion. Pump failure leads to intracellular water molecules becoming trapped within the cells from loss of the concentration gradient across the cell membrane. The result is restriction in water molecule movement both across the membrane due to loss of gradient, as well as reduction in space between the edematous cells for water movement to occur.[25,34,35]

DWI sequences can measure these restrictions in water diffusion. This sequence relies on MR techniques, which are beyond the scope of this article but the resultant images prove to be highly useful in the detection of pathologic conditions where diffusion restriction is a common characteristic. Areas where diffusion occurs freely will appear dark whereas regions of restrict diffusion will appear bright. Early detection of ischemic stroke is made possible with DWI as it is able to detect the presence of diffusion restrictions within the initial minutes to hours following onset of symptoms.[25,35–37]

DWI when used with apparent diffusion coefficient mapping more readily detects acute ischemic stroke, particularly in those presenting shortly after symptom onset or a small core infarct (**Fig. 12**).

MRI has its own unique benefits and limitations, which need to be considered when selecting an appropriate imaging modality (**Table 2**). On the one hand, it avoids ionizing radiation, can allow for accurate vascular imaging of the CNS without the use of contrast agents in those who are sensitive or at risk for kidney injury. Additionally, it often has superior imaging resolution, which may be necessary in making a diagnosis. On the other hand, it is often more time consuming to obtain compared to CT imaging. Patients with metallic implants, pacemakers, or retained foreign objects may be ineligible for MRI due to risk of injury and/or device failure due to magnetic incompatibility. Additionally, patients with claustrophobia, those who are restless and cannot remain still, may not be able to undergo imaging without sedation to facilitate.

When choosing to pursue emergent neuroimaging in the ED, beware that the easiest test may not always be the best or most appropriate choice. For example, NCCT can be falsely reassuring in patients with signs and symptoms of ischemic stroke or

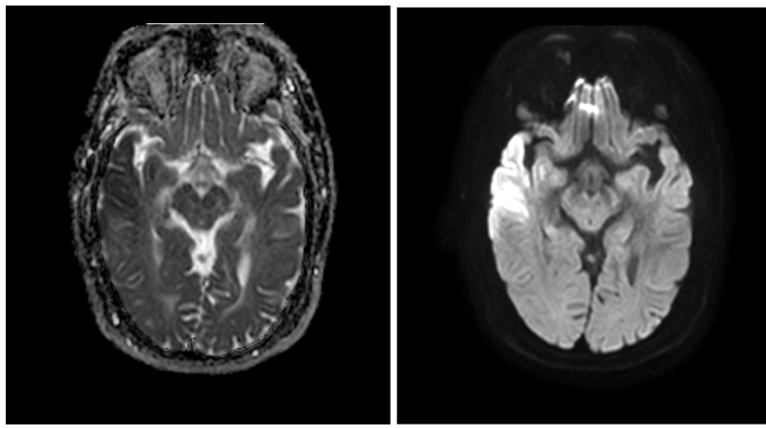

Fig. 12. Left: Diffusion imaging with apparent diffusion coefficient mapping of left MCA territory infarct Right: DWI sequence of right MCA infarction.

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Table 2
Pearls and pitfalls to effective neuroimaging utilization in the emergency department

MRI Sequence	Appearance of Key Structures	Most Useful For	Image
T1	Fat/White Matter: Bright Gray Matter: Dark CSF: Black	• Delineating anatomic details • Identifying atrophy & structural abnormalities associated with neurodegenerative conditions ○ Examples ■ Alzheimer's Disease–> hippocampal atrophy ■ Frontotemporal dementia–> Frontal and temporal atrophy	

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Table 2
(*continued*)

MRI Sequence	Appearance of Key Structures	Most Useful For	Image
T2	Fat/White Matter: Dark Gray Matter: Bright CSF: White Tissues with higher water content (eg, due to edema or swelling) appear bright	• Identifying conditions which cause tissue edema ○ Stroke ○ Tumor ○ Infections ○ Multiple Sclerosis	

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T2 & SWI	Hemorrhage: Dark spots Calcifications/Iron deposits: Dark spots Other Tissues: Variable	• Identifying ○ Hemorrhage ○ Vascular Malformations ○ Calcifications (eg, within tumors or vascular malformations)	

(*continued on next page*)

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Table 2
(*continued*)

MRI Sequence	Appearance of Key Structures	Most Useful For	Image
FLAIR (Fluid Attenuated Inversion Recovery)	White Matter: Dark Gray Matter: Bright CSF: Black Periventricular lesions will typically appear bright	• Identifying ○ Periventricular lesions (eg, tumors, cystic lesions, MS near verticles) ○ SAH ○ Meningitis, Encephalitis (consider contrast which can improve sensitivity/ specificity)	

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DWI (Diffusion Weighted Imaging)	Healthy Tissue: Dark Ischemic Tissue: Bright Edematous Areas: Bright	• Identifying ○ Acute ischemic stroke ■ Appears bright, minutes to hours after symptom onset ○ Dai ■ Bright signals in white matter due to axonal swelling, within hours of injury ○ Hie ■ Bright spots white matter, basal ganglia, thalamus from cytotoxic edema.	
MRA (Magnetic Resonance Angiography, Non-Contrast)	Vasculature: Bright Tissues: Dark	• Delineating vascular anatomy • Identifying ○ Large Vessel Occlusions (LVOs) ○ AV malformations ○ Aneurysmal Disease	

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transient ischemic attack and current ACEP guidelines suggest CTA and/or MRI when feasible.[38] On the other hand, patients presenting with acute, uncomplicated headache without red flag features to suggest dangerous pathology should likely not undergo any neuroimaging at all.[39] Clinicians may be tempted to image patients with vague neurologic complaints, and while the gestalt in knowing who to image is a key, choosing the appropriate imaging modality is just as important. Dizziness, as an example, is a common ED chief complaint associated with high rates of diagnostic error.[40] Dizzy patients diagnosed with peripheral vertigo who underwent NCCT have been shown to portend higher rates of short-term stroke than patients not imaged at all suggesting clinicians identified a high-risk cohort, but were falsely reassured by an imaging modality that is poorly sensitive for stroke.[41] The Society of Academic Emergency Medicine Guidelines for Reasonable and Appropriate Care in the Emergency Department (SAEM GRACE-3) program provides a comprehensive approach to the dizzy patient in the ED including appropriate neuroimaging selection and utilization.[42] As a general rule, when time sensitive treatment decisions are needed, the speed and efficiency of NCCT (sometimes bundled with CT angiography) is the best approach, such as in suspected acute ischemic stroke within a reperfusion window. When a more nuanced approach is indicated, deferring the swiftness of NCCT in favor of MRI may be more appropriate and ultimately a better use of resources.

As the neuroimaging field continues to evolve, undoubtedly there will continue to be advancements in the role of artificial intelligence in image acquisition and interpretation. Machine learning technologies can potentially reduce scan time and improve timely detection of diagnoses, such as LVO stroke. While AI applications can be harnessed to improve timely care delivery, they also have the potential to be misused or over-relied upon, introducing the potential for automation bias.[43] EM clinicians should work closely with their colleagues in radiology and neurologic sciences to ensure shared understanding of their applications in each system.

CLINICS CARE POINTS

- Use a systematic approach such as 'Blood can Be Very Bad' mnemonic when evaluating CT scans of the brain for pathology.
- Non-contrast CT scan is the initial imaging modality of choice when evaluating for spontaneous and traumatic intracranial bleeding.
- CT or MR angiography should be used when assesing for vessel occlusion in ischemic stroke, dissection, and vasospasm.
- When reviewing an MRI for suspected ischemic stroke, use the DWI sequence to assess for cytotoxic edema (will appear bright and correspondingly dark on the Apparent Diffusion Coefficient sequence), the T2 FLAIR sequence to assess for vasogenic edema, and the SWI (or grandienc echo) sequence to assess for hemorrhage.
- Contrast-enhanced CT and MR are important when assessing for infectious and inflammatory conditions.

DISCLOSURE

The authors declare that there are no financial disclosures related to the article.

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